[PS1.53]

silCR specific antibodies increase infection severity in a mouse model of necrotizing fasciitis caused by group G streptococcus

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Introduction: Group G streptococci (GGS) cause severe invasive infection in patients mostly with co-morbidities. GGS possess virulence factors similar to the more virulent group A streptococcus (GAS). There is an increased frequency of reports on human infections caused by GGS.

a streptococcal invasion locus, sil, was identified in GAS isolated from a patient with necrotizing fasciitis (NF). sil is highly homologous to a regulon of *Streptococcus pneumoniae* involved in bacterial signaling. *sil*CR encodes a putative competence-stimulating peptide (CSP).

82% of GGS possess sil while only 24% of GAS. The high prevalence of sil in less virulent GGS and the correlation in GAS between presence of sil and less severe invasive infection, suggests that streptococci harbouring sil may be less virulent.

Methods: silCR was detected on bacterial surface and in growth medium by immunoblot using anti-silCR antibodies, confirmed by immune-fluorescence.

A modified-NF-model using cyclophosphamide was used to assess mice protection injecting bacteria grown with the synthetic silCR.

Mice were vaccinated with silCR and tested for antibody production. silCR vaccinated mice were compared to non-vaccinated mice in their response to a NF infection by GGS.

Results: silCR was identified on GGS surface and is secreted to the growth medium.

Mice injected with GGS grown in the presence of synthetic silCR were protected from infection.

Mice vaccinated with silCR produced anti-silCR antibodies. Vaccinated mice developed a significantly more severe infection compared to non-vaccinated mice.

Conclusion: Our results indicate that GGS strains that harbor sil express and secrete the silCR peptide similar to what is known for quorum sensing peptides.

We confirmed that the silCR peptide confers protection against a NF infection. Furthermore, production of anti-silCR antibodies renders mice more susceptible to severe infection caused by GGS. Antibodies produced against bacterial determinants are in fact detrimental to mice in this model of infection.

Keywords: infection, antibodies, peptide, bacteria